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Hemorrhagic Arachnoid Cyst with Third Nerve Paresis: CT and MR Findings

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Summary: We report the CT and MR appearance of a nontraumatic hemorrhagic arachnoid cyst presenting with a third nerve paresis in a 37-year-old man. The cyst, located in the left suprasellar area, contained a fluid-blood level with stigmata of subacute hemorrhage on both CT and MR studies.

Index terms: Arachnoid, cysts; Cerebral hemorrhage; Nerves, oculomotor (III)

Arachnoid cysts account for approximately 1% of intracranial space-occupying lesions (1). Complications are rare, and clinical presentation depends on their location. We describe the computed tomographic (CT) and magnetic resonance (MR) imaging features of a suprasellar arachnoid cyst complicated by an isolated intracystic hemorrhage with an unusual clinical manifestation.

Case Report

A 37-year-old man presented with a headache of sudden onset and 2 days' duration, followed by diplopia and blurred vision of the left eye. He had no history of head trauma. Results of a physical examination were normal except for a paresis of the third left nerve and a left mydriasis.

A noncontrast CT scan showed an oval hyperdense fluid-blood level in the suprasellar area (Fig 1A). MR imaging, performed immediately after CT, confirmed the presence of an ovoid well-defined cystic lesion containing a fluid-blood level in the subarachnoid space above the sella turcica in front of the left cerebral peduncle. The top layer of the lesion had high signal intensity on T1-weighted spin-echo images (Fig 1B) and very high signal intensity on proton density- and T2-weighted turbo spin-echo images (Fig 1C and D). The dependent layer had an intermediate signal on T1-weighted spin-echo images and on proton density-weighted (Fig 1C) and T2-weighted (Fig 1D) turbo spin-echo images. The mass did not enhance

after intravenous injection of gadopentetate dimeglumine (Fig 1E and F). It measured $1.8 \times 1.6 \times 1.1$ cm, and was located extraaxially in the opticochiasmatic and interpeduncular cisterns along the course of the left third cranial nerve. It displaced the left optic tract upward (Fig 1E and F) and effaced the left amygdala outward (Fig 1E). The posterior communicating arteries, the left one in particular, were not seen on MR angiograms of the circle of Willis or on conventional cerebral angiograms obtained before surgery.

Laboratory coagulation tests (prothrombin time, partial thromboplastin time, and fibrinogen) and platelet level were normal. The patient had no pertinent medical history (liver disease or renal dialysis) and had not taken any medication that could have caused hemorrhage. Surgery performed 3 days after admission consisted of draining and completely excising the cystic wall after carefully dissecting the third left nerve that was closely adherent to it. The cyst contained dark brown fluid and showed evidence of recent hemorrhage. Pathologic examination confirmed the diagnosis of a hemorrhagic arachnoid cyst: the cyst wall was lined by meningoendothelial cells (Fig 1G) that were, as expected, positive for epithelial membrane antigen (EMA) by immunolabeling (Fig 1H) and cells containing hemosiderin (Fig 1G), as confirmed by their positivity at Perls staining (Fig 1I). The patient made a good postoperative recovery but still had complete palsy of the third left nerve on the 12th postoperative day.

Discussion

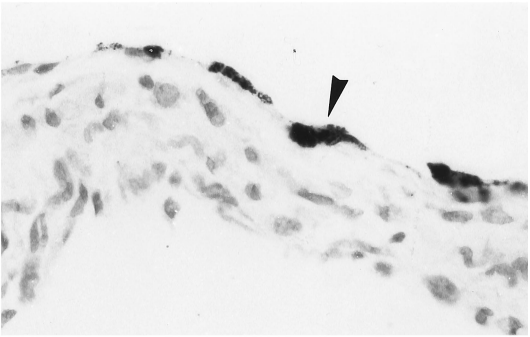
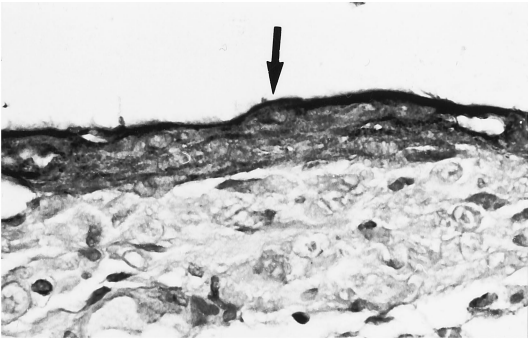
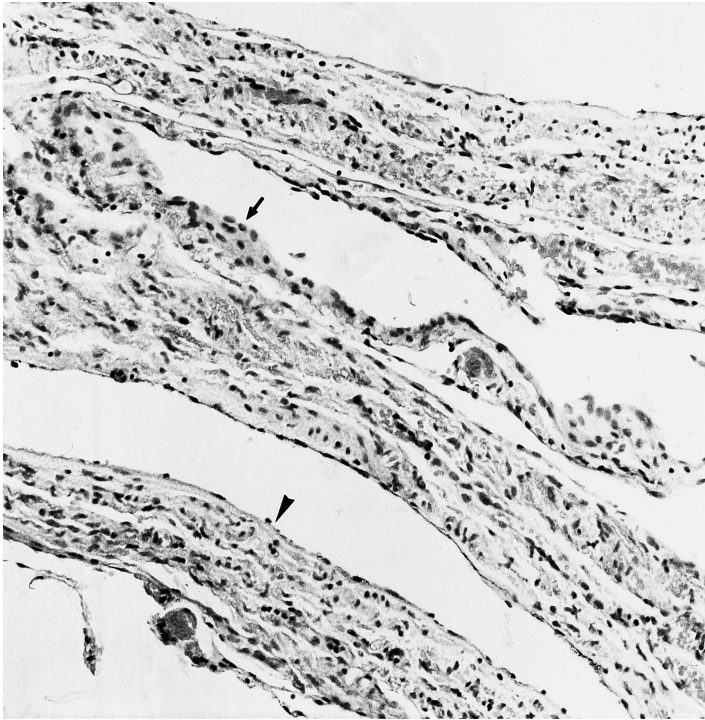
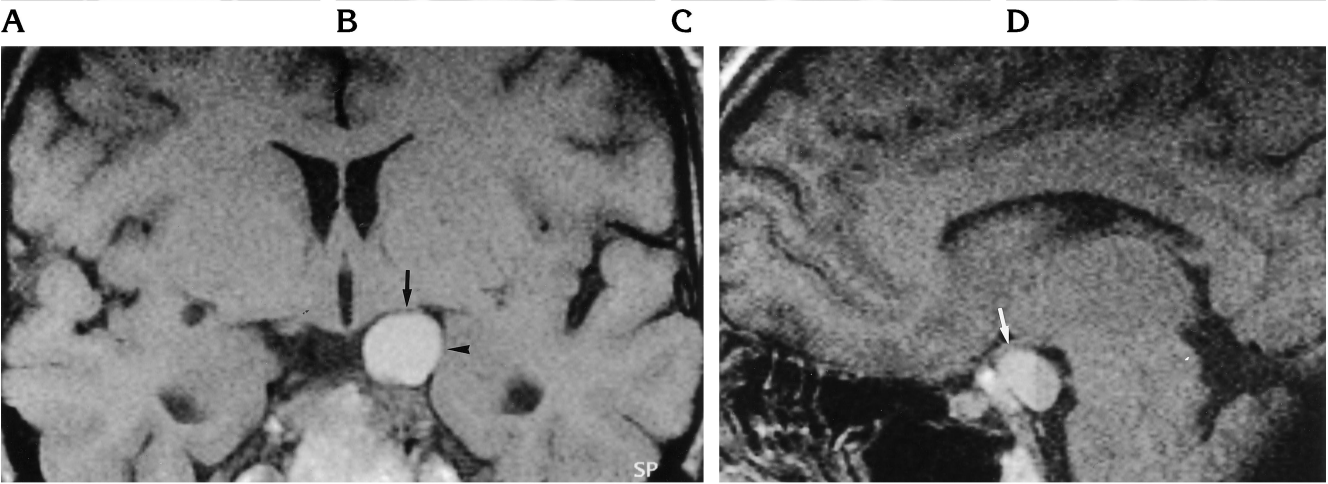
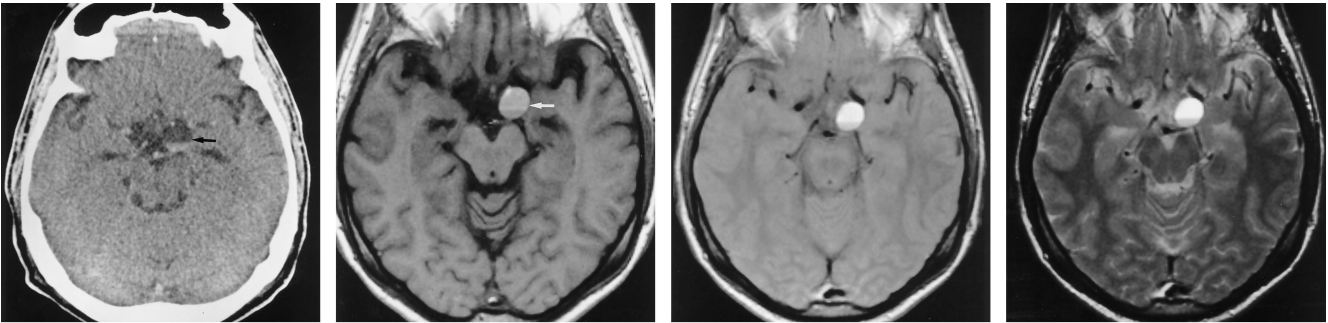
Arachnoid cysts are benign, congenital, intraarachnoidal space-occupying lesions that are filled with a clear fluid, similar to cerebrospinal fluid (CSF) (2). With the advent of CT and MR imaging, they have been increasingly recognized (3). Their development is probably the result of splitting or duplication of the arachnoid membrane (4). Common locations are the middle cranial fossa, the suprasellar region, as

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in our case, the quadrigeminal region, the cerebral convexity, the interhemispheric fissure, the cerebellopontine angles, and the cisterna magna (5). They are usually asymptomatic, but may at times act as a mass lesion with compressive effect due to size or hemorrhage, and present with headache, nausea, vomiting, confusion, or other signs and symptoms, depending on their location.

Suprasellar arachnoid cysts have been associated with Kallman syndrome (6), precocious puberty (7, 8), and involvement of the optic nerve and chiasm (9, 10). Although rare, cranial nerve palsy in relation to arachnoid cysts has been reported at other sites. Hemifacial spasm caused by an arachnoid cyst located in the cerebellopontine angle has been reported by Higashi et al (11) and by Altinors et al (12). Cartwright et al (13) reported a posterior fossa arachnoid cyst presenting with an isolated 12th nerve paresis (13). An arachnoid cyst of the cerebellopontine angle manifesting as contralateral trigeminal neuralgia was reported by Babu and Murali (14).

Isolated intracystic hemorrhage represents a rare but recognized complication of arachnoid cysts and may be the initial clinical presentation (3, 15). It may be spontaneous, due to minor trauma with rupture of intracystic or bridging vessels (3), or it may follow more severe trauma (16). Recently, Hirose et al (17) reported an intracystic hematoma without subarachnoid hemorrhage, which was caused by rupture of an aneurysm into an arachnoid cyst. After trauma, arachnoid cysts of the middle cranial fossa are sometimes associated with subdural hematoma (18–21) and, rarely, with extradural hematoma (22). In our case, hemorrhage was spontaneous and confined strictly to the cystic cavity.

Both CT and MR studies showed the presence

of intracystic hemorrhage, but the MR images better depicted the anatomic relationships before surgery (Fig 1E and F). The hemorrhagic pattern consisted of a horizontal interface separating the fluid into two layers. This pattern is the result of sedimentation of red blood cells into a cavity previously filled with clear fluid that resembles CSF. The main intrinsic factors influencing the hemorrhagic signal in our case were not only the specific form of hemoglobin present and the status of the red blood cells but also the presence of CSF-like fluid in the hemorrhagic site. The top layer consisted of lysed red blood cells and proteinaceous serosanguineous serum. Combined effects of dilute-free methemoglobin (short T1, long T2) and free water (long T1, long T2) may have accounted for the high signal intensity on T1-weighted images and the very high signal intensity on T2-weighted images. The dependent layer consisted of intact red blood cells containing methemoglobin. Intermediate signal intensity on both T1- and T2-weighted images was thought to be due to the opposed effects of intracellular methemoglobin (short T1, short T2) and high water content (long T1, long T2). This is certainly a simplified explanation for the lesion signal, as the MR appearance of hemorrhage is complex and depends on other intrinsic and extrinsic factors. For example, the turbo spin-echo technique, which is known to be less sensitive to magnetic susceptibility, is an extrinsic factor that may have affected the hemorrhagic signal on T2-weighted images (23). On CT scans, hyperattenuation of the dependent layer is due to higher hemoglobin concentration.

Such blood-fluid levels are a common finding in intraventricular bleeding and in recurrent hemorrhage in preexisting chronic subdural he-

Fig 1. Arachnoid cyst complicated by intracystic hemorrhage in a 37-year-old man.

A–D, Unenhanced CT scan (A), transverse unenhanced T1-weighted spin-echo MR image (600/14/2 [repetition time/echo time/excitations]) (B), and transverse proton density-weighted (2500/14/2) (C) and T2-weighted (2500/85/2) (D) turbo spin-echo MR images at the same level as A show an oval cystic lesion in the left part of the suprasellar cistern, containing a fluid-blood level (arrow in A and B). The upper layer, consisting of serum with extracellular dilute-free methemoglobin, was hypodense on the CT scan, hyperintense on the T1-weighted MR image, and very hyperintense on the proton density- and T2-weighted images. The dependent layer of the lesion, consisting of intact red blood cells containing methemoglobin, appears hyperdense on the CT scan and has intermediate signal intensity on the MR images.

E and F, Contrast-enhanced coronal (620/20/2) (E) and sagittal (390/20/2) (F) T1-weighted spin-echo MR images show absence of enhancement and superior displacement of the left optic tract by the cyst (arrows). On the coronal image, note effacement of the left amygdala (arrowhead).

G–I, The cyst is lined by meningoendothelial cells (arrow, G; hematoxylin-eosin, original magnification $\times 120$), as confirmed by their immunopositivity for EMA (arrow, H; immunolabeling for EMA, original magnification $\times 360$) and by cells containing hemosiderin resulting from a previous hemorrhage (arrowhead, G), as determined by positivity for Perls staining (arrowhead, I) (original magnification $\times 60$).

matoma; they have also been reported in intracerebral parenchymal hemorrhage in association with coagulopathy (24), amyloid angiopathy (25), tumor (24), and radiation necrosis (26).

The differential diagnosis of suprasellar cystic masses likely to bleed includes nonneoplastic cysts, such as Rathke's cleft cysts and neuroglial cysts. Parasitic cysts are an additional consideration, especially in endemic regions. It is usually possible to distinguish cystic tumors, either primitive or secondary, on the basis of their enhancement characteristics and their solid tissue component with eventual calcifications. In our opinion, the possibility of an aneurysm of the posterior communicating artery, a common lesion in this location, could be disregarded owing to the presence of a blood-fluid level.

In conclusion, hemorrhage in an arachnoid cyst can present radiologically with a blood-fluid level and clinically with a third nerve paresis.

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